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Traumatic Haemoabdomen

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SUMMARY

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Haemoabdomen is an important differential diagnosis for canine and feline abdominal trauma. The diagnosis is made by aspiration of blood from the abdomen by abdominocentesis. Spleen and liver are the most likely sources of traumatic bleeding. Patients are stabilized with appropriate fluid therapy, oxygen supplementation and analgesia. With ongoing haemorrhage, serial measurement of abdominal and venous haematocrit can be helpful in making the decision between surgical and medical therapy. Most patients with traumatic haemoabdomen can be treated medically. Surgical therapy should be reserved for patients that cannot be stabilized despite medical intervention. The surgical approach should be thoroughly planned in order to minimize further abdominal blood loss and blood transfusions should be readily available.

Key words: haemoperitoneum, dog, cat, diagnostic procedures

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Introduction

Haemabdomen or haemoperitoneum describes the pathologic accumulation of blood in the abdomen. Causes of haemoabdomen can be traumatic or non-traumatic. Blunt abdominal trauma is common in trauma patients, but the prevalence of traumatic abdominal bleeding is unknown and depending on the severity, haemoabdomen can be clinically nonsignificant.

The diagnosis of haemoabdomen necessitates the differentiation of active versus inactive bleeding which is often difficult. However, immediate diagnosis and adequate stabilisation are mandatory for the successful treatment of patients with abdominal bleeding. The treatment of haemoabdomen includes emergency surgery as well as medical therapy [Mongil *et al.*, 1995; Sigrist and Spreng, 2007].

Case discussion

A two-year old male Labrador is presented 30 minutes after being hit by a car. At presentation, primary survey revealed lethargy, tachycardia (180 beats per minute), pale mucous membranes and a prolonged capillary refill time (CRT) of 2 seconds. Respiratory rate was 40 breaths per minute and bilaterally increased lung sounds could be auscultated. Abdominal palpation was painful and the clinician noticed a possible fluid wave. Orthopaedic and neurologic examinations were within normal limits. The initial CBC and biochemical profile showed a venous PCV of 44 %, total solids (TS) of 50 g/l (reference 57-75 g/l), normal leucocyte and thrombocyte numbers, albumin 25 g/l (normal 27-38 g/l), creatinine 100 µmol/l (reference 53-120 µmol/l), ALT 6306 IU (reference 24-124 IU), AST 8284 IU (reference 20-73 IU), GLDH 268 IU (reference 2-10 IU) and normal electrolytes. PT was 14.6 seconds (s) (reference 6.3-8.5 s) and aPTT 8.6 s (reference 9.6-16.1 s). Indirect systolic blood pressure was 128 mmHg with a mean arterial blood pressure (MAP) of 78 mmHg. Simultaneously with the venous blood collection the abdomen

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was tapped. Serosanguinous fluid was aspirated. Abdominal fluid showed a PCV of 42 %, TS of 43 g/l and creatinine of 96 µmol/l. A presumptive diagnosis of haemoabdomen was based on these results.
Stabilization of the dog included nasal oxygen supplementation, a bolus of 10 ml/kg Plasmalyte® (Baxter AG) as well as 5 ml/kg hydroxyethyl starch (Voluven®, Fresenius Kabi AG), followed by lactated Ringer's solution (Fresenius Kabi AG) at 2ml/kg/h, Voluven® at 1 ml/kg/h, amoxicillin-clavulanic acid (Augmentin®, GlaxoSmithKline, 20 mg/kg i. v. q 8 hours), analgesia [initially methadone (Methadon Streuli®, Streuli Pharma AG) 0.1 mg/kg i. v. q 2 hours followed by buprenorphine (Temgesic®, Essex Chemie AG) 0.01 mg/kg i. v. q 8 hours]. Monitoring included hourly measurement of heart rate, respiratory rate, mucous membrane color, CRT and blood pressure.

On chest radiographs, the dog showed lung contusions and a small pneumothorax. Ultrasonic examination of the abdomen revealed large amounts of free fluid, signs of liver rupture with haematoma formation, evidence of a blood clot in the bladder and signs of pancreatic oedema and/or haematoma.

Two hours after presentation the venous PCV had decreased to 26 %. The abdominal PCV measured at the same time was 40 %. Four hours after presentation, the dog showed a venous PCV of 22 % while the abdominal PCV was 41 %. Eight hours later the venous PCV stabilized at 23 % and concurrent abdominal PCV was 40 %. The dog remained haemodynamically stable. Venous PCV increased to 25 % the following day and normalized within a couple of days. The dog remained stable and went home 3 days later without further diagnostic or therapeutic procedures.

Aetiology

Traumatic haemoabdomen is a potentially live-threatening complication of blunt or penetrating abdominal trauma and is most often seen after motor vehicle accidents. The spleen and liver have been described as common sources of haemorrhage in human as well as veterinary patients [Clarke *et al.*, 2002; McKenney *et al.*, 2001]. Due to its location and fragility, the liver is the most common source of haemorrhage.

Physical examination

Clinical symptoms may vary from patient to patient and depend on other trauma-related abnormalities such as shock, respiratory distress and other abdominal or extra-abdominal injuries. After initial stabilisation of respiration and perfusion, a complete physical exam is indicated in all patients. Clinical signs of abdominal bleeding may appear several hours after trauma. Depending on the amount of blood loss, signs of hypovolaemic shock such as pale mucous membranes (MM), prolonged capillary refill time, tachycardia and a weak femoral pulse as described in the above case description may be seen. During the compensation phase of hypovolaemic shock, animals may present with red mucous membranes and a short capillary refill time [de Laforcade 2008]. Abdominal bleeding as well as other trauma-related injuries will lead to pain and patients will present with signs of an acute abdomen and pain on abdominal palpation. Depending on the amount of fluid accumulation and body configuration a fluid wave may be palpable. At least 40 ml/kg of fluid is needed for a clear fluid wave to occur on abdominal palpation [Crowe and Devey, 1994]. Occasionally umbilical and peri-testicular skin discoloration may be observed when significant intra-abdominal haemorrhage dissects through the abdominal muscle planes and subcutis [Crowe and Todoroff, 1982]. A normal physical exam does not exclude traumatic haemoabdomen since haemoabdomen, as well as other traumatic injuries to the abdomen may not be recognized reliably [Crowe and Crane 1976; Davies *et al.*, 1976].

Differential Diagnosis

Differential diagnoses for traumatic haemoabdomen include all other traumatic injuries such as bladder or urethral rupture, peritonitis following intestinal or bile duct injuries, traumatic pancreatitis or traumatic shock [Crane 1980]. These trauma-related injuries might also be seen in addition to traumatic haemoabdomen and have to be excluded during diagnostic workup of the patient. Causes of non-trauma-related haemoabdomen include neoplasia, coagulopathy and splenic or liver torsion. Other differential diagnoses in patients presenting with signs of an acute abdomen are peritonitis, intestinal obstruction, pancreatitis or neoplasia.

Diagnostic approach

In human medicine, the diagnostic evaluation of blunt abdominal trauma combines clinical evaluation, abdominal ultrasound, computed tomography (CT) evaluation and analysis of abdominal effusion retrieved by abdominocentesis or diagnostic peritoneal lavage (DPL) [Hoff *et al.*, 2002].

Laboratory evaluation

Acute bleeding leads to a similar loss of erythrocytes and plasma. PCV and total solids (TS) will therefore not change in the first few minutes following trauma. The decreased intravascular volume then leads to splenic contraction with subsequent release of sequestered red blood cells (dog >> cat) and to a shift of water from the interstitial into the intravascular compartment, leading to a lower TS compared to the PCV value. PCV may therefore be normal with acute haemorrhage. A significant decrease in PCV and TS values may be seen only after fluid therapy directed at normalizing intravascular volume [Crane 1980]. Regular PCV checks are therefore mandatory during resuscitation. Blood gas analysis and lactate measurements may help in interpretation of perfusion status, with metabolic acidosis and hyperlactataemia being suggestive of cellular hypoxia [de Laforcade and Silverstein, 2008]. Increases in liver enzyme activities may commonly be seen after trauma and are not associated with the cause of abdominal haemorrhage. Additional laboratory evaluation including complete blood count, biochemical profile and urinalysis is often normal but may be helpful in excluding other concurrent diseases or trauma-related organ dysfunction. Depending on the history and physical exam results, coagulopathy as a cause of non-trauma-related haemoabdomen should be ruled out.

Diagnostic imaging

Abdominal radiographs in the presence of abdominal effusion are of questionable diagnostic value, as the free abdominal fluid will lead to decreased abdominal detail. Abdominal radiographs may though be helpful to exclude severe haemoabdomen in trauma patients and may detect other trauma-related injuries [Brockmann, 2000]. Thoracic radiographs may be indicated in trauma patients in order to identify or exclude pneumothorax, haemothorax, diaphragmatic rupture or rib fractures that would require additional treatment. Abdominal ultrasound is helpful in the identification of abdominal effusion and possibly in the identification of its cause [McKenney *et al.*, 1998]. Scanning defined abdominal locations using "focused assessment with sonography for trauma" (FAST) shows a high sensitivity and specifity for the diagnosis of haemoabdomen in trauma patients [Scalea *et al.*, 1999; von Künsberg *et al.*, 2003]. FAST, used as a scoring method, may help to estimate the extent of abdominal effusion and may be used as a parameter to decide if a surgical approach is required. A positive FAST result in haemodynamically unstable human patients is an indication for laparotomy, while a positive FAST result in stable patients requires CT evaluation in order to identify the cause of abdominal haemorrhage [Scalea *et al.*, 1999]. In human studies, the specifity of this method is 71-78% [Ma *et al.*, 1995; Huang *et al.*, 1994].

A FAST protocol has been developed for dogs and showed a sensitivity of 96% and a specifity of 100% in terms of identification of an abdominal effusion [Boysen *et al.*, 2004]. With the patient in left lateral recumbency, the abdomen is screened for free abdominal fluid at the following locations: immediately caudal to the xiphoid process, on the ventral midline over the bladder, over the right flank (gravity-independent region) and over the most gravity-dependent area of the left flank [Boysen *et al.*, 2004]. The presence of abdominal fluid is not specific for the diagnosis of haemoabdomen however and identification has to be followed by abdominocentesis and fluid analysis. Computed Tomography is the standard diagnostic and monitoring tool used in human patients presenting with haemoabdomen [Baron *et al.*, 1993]. In veterinary medicine, CT imaging is not widely available and the advantage over DPL remains controversial in human medicine [Blow *et al.*, 1998].

Abdominocentesis

Any finding or suspicion of free abdominal fluid accumulation should prompt abdominocentesis and fluid analysis. Abdominocentesis is a sterile procedure and is most simply done under ultrasound guidance using an 18-22 G hypodermic needle with attached syringe. Blind paracentesis following the 4-quadrant rule is another option (figure 1). A hypodermic needle with or without an attached syringe is introduced into the abdomen paramedian either cranially or caudally to the umbilicus [Walters, 2003]. Depending on abdominal palpation results (cranial organomegaly versus large bladder), initial paracentesis is done either caudally or cranially to the umbilicus at the most gravity-dependent location. If no fluid is retrieved, another quadrant is tried. Using the aspiration technique, omentum may occlude the needle, requiring open paracentesis and collection of fluid from the needle hub [Walters, 2003]. The accuracy of abdominocentesis for detection of haemoabdomen is 50-62% [Crowe and Crane, 1976; Kolata *et al.*, 1976]. The use of a peritoneal dialysis catheter increases the sensitivity up to 100 % [Crowe and Crane, 1976; Crowe, 1984]. False-positive results may be seen after aspiration of liver, spleen or abdominal vessels [Crowe and Bjorling, 1993]. This can easily be ruled out if the aspirated blood does not clot, as blood in contact with abdominal serosa is depleted of fibrinogen and thrombocytes [Prasse and Duncan, 1976].
Diagnostic peritoneal lavage (DPL) can be performed when

FIGURE 1A and 1B: Abdominocentesis using 4-quadrant aspiration.
The abdomen is punctured para¬median 2-3 cm from the midline. Depending on the sus¬pected disorder, the first puncture is done cranially or cau¬dally to the umbilicus (figure 1A, "X"). The puncture is repeated in one quadrant after the other, until fluid can be retrieved.
Aspiration is best done using an 18-22 G hypodermic needle attached to a 5-10 cc syringe after surgical preparation of the site. If no fluid can be aspirated, puncture is tried without aspiration.

Figure 1A

Figure 1B

paracentesis techniques do not provide a positive diagnosis and ultrasound is not available [Crowe, 1984]. Warm sterile 0.9% saline is infused into the peritoneal cavity through the DPL catheter (20 ml/kg) and the fluid is allowed to mix with fluid present in the abdominal cavity for 15 minutes. The fluid is collected by gravity flow into a sterile closed collection system and analyzed. DPL allows the identification of as little as 0.8 ml/kg blood [Crowe and Crane, 1976]. PCV values higher than 2-5% in DPL fluid are associated with severe haemoabdomen [Crowe, 1993; Dye, 1999]. Unfortunately, neither abdominocentesis nor DPL allow differentiation between active and inactive abdominal bleeding [Bilge and Sahin, 1991]. With increased availability of sonography, DPL has been largely replaced by ultrasound-guided abdominocentesis.

Paracentesis fluid is collected into EDTA, heparin and sterile serum tubes. EDTA-fluid is used for the determination of PCV/TS and cell count and for cytological evaluation. Creatinine or bilirubin can be determined from heparinized blood if indicated. If the PCV of the abdominal fluid matches the venous PCV or is even higher, the diagnosis of a haemoabdomen can be made. Abdominal fluid with a lower than venous but still substantially high PCV is indicative of abdominal bleeding in combination with another effusion and may necessitate measurement of abdominal and venous creatinine to rule out uroabdomen. Higher creatinine concentrations in the abdominal fluid are indicative of uroabdomen [Schmeidt *et al.*, 2001]. According to this approach, an increased abdominal bilirubin concentration is indicative of bile peritonitis. Cytology is used to rule out septic peritonitis [Conally, 2003].

Therapy

Depending on the cause of haemoabdomen, patients are treated surgically or non-surgically after initial stabilization [Hoff *et al.*, 2002; Nagy *et al.*, 1995]. Severe abdominal haemorrhage that needs immediate surgical intervention should be suspected in patients that cannot be stabilized. Smaller abdominal bleeding is usually self-limiting and patients may not show any clinical symptoms. In any case, the patient must be stabilized prior to any diagnostic or surgical interventions.

Stabilization

Stabilization of a patient with haemoabdomen follows the ABC. Fluid therapy and analgesia are important therapeutic interventions in all patients, regardless of medical or surgical management.

Fluid therapy: Patients with abdominal bleeding will present in hypovolaemic shock. Aggressive treatment of hypovolaemic shock increases survival [Gallerani-Santibanez *et al.*, 2001]. Fluid therapy therefore is an important step in the stabilization of the patient and aims to normalize tissue perfusion and oxygen delivery while minimizing further bleeding. A combination of isotonic crystalloids and colloids is advised [Sigrist, 2005]. With haemorrhage, there is a risk of further bleeding with normalization of perfusion. If blood pressure is increased substantially prior to definitive haemostasis, further haemorrhage may occur [Revell *et al.*, 2003; Prough *et al.*, 1991]. On the other hand, attention must be given to maintaining an adequate blood pressure until haemostasis has occurred. The concept of "low volume resuscitation" is accomplished by maintaining a mean arterial blood pressure (MAP) of 60 mmHg [Sondeen *et al.*, 2003]. Fluid therapy is carried out with intravenous boluses of 10-20 ml/kg of an isotonic crystalloid fluid such as 0.9% NaCl or lactated Ringer's solution and 5-10 ml/kg of a colloid such as hydroxyethyl starch (Voluven®). Hypertonic saline (4 ml/kg) together with smaller boluses of crystalloids and colloids would be another option [Varicoda *et al.*, 2003]. The volume expansion with hypertonic saline may be difficult to control and hypertonic saline should therefore be reserved for severely hypotensive animals. Hypotonic solutions such as DW5 or glucose-NaCl combinations should not be used in hypovolaemic shock. With severe haemorrhage, whole blood transfusions may be necessary in order to maintain oxygen carrying capacity and haemostasis.

Oxygenation: Oxygen supplementation is indicated for hypovolaemic shock as well as additional lung dysfunction in trauma patients. Oxygen supplementation during the stabilization process is easiest achieved using a mask or flow-by oxygen. Should continued oxygen supplementation be required, a nasal oxygen catheter can be placed and oxygen supplied at 2-5 l/min. Arterial oxygen saturation should be at least 95% in order to ensure sufficient tissue oxygenation. Anaemic patients should have a higher saturation (SpO2 99-100 %) and maintaining sufficient oxygenation may require transfusion therapy.

Transfusion therapy: Loss of oxygen-carrying erythrocytes will lead to tissue hypoxia. Hypovolaemia and resulting hypoperfusion will increase this effect. There is no real transfusion trigger [Day, 2000; Jutkowitz, 2004]. The decision to administer a red blood cell transfusion or a blood substitute solution (Oxyglobin®) should be considered when clinical signs compatible with severe anaemia and haemorrhagic shock (tachycardia, tachypnoea, bounding pulses, collapse) are seen, when required fluid therapy will lead to a declining PCV or when PCV declines below 20-25% [Herold *et al.*, 2008; Jutkowitz, 2004]. The choice of blood product depends on availability and the presence of coagulation factor deficiencies. With severe haemorrhage, loss and dilution of erythrocytes as well as coagulation factors may require transfusion of fresh whole blood. The whole blood transfusion must be fresh (<6 hours) in order to provide coagulation factors. Isolated anaemia with normal coagulation times can be treated with a pRBC transfusion and coagulation factor deficiencies leading to haemoabdomen but only mild anaemia may be treated with plasma alone. Detailed descriptions of transfusion therapy are available elsewhere [Sigrist, 2005, Hohenhaus, 2000].

Autotransfusion: Catastrophic abdominal bleeding may require autotransfusion of shed abdominal blood. Autotransfused blood may be live-saving as it is readily available and presents no risk of transfusion reaction. The abdominal blood is collected aseptically and mixed with an anticoagulant solution [Crowe 1980, Jutkowitz, 2004]. Anticoagulation is carried out with sodium citrate 3.8% (ratio 1:9) or CPDA (ratio 1:7). Blood can also be collected directly into commercially available blood collection bags. The advantages of autotransfusion must be

evaluated in light of potential side effects such as potential induction of disseminated intravascular coagulation by cell debris and transfusion of bacteria from potential abdominal contamination. Autotransfusion is contraindicated if abdominal blood is contaminated with tumor cells, urine or bacteria from gastrointestinal rupture [Crowe 1980]. The authors recommend autotransfusion only if insufficient blood products are available. All blood products are administered using a filter.

Analgesia: NSAID's are contraindicated in hypovolaemic animals with decreased renal perfusion; therefore opioids are the analgesics of choice in patients with haemoabdomen. Short-acting, pure μ-agonists such as fentanyl (2-10 μg/kg/h) or methadone (0.1-0.2 mg/kg q 1-2 h) are preferred as the dose can be adjusted and they can be antagonized if necessary. Intravenous lidocaine has analgesic and antioxidant properties and is a good choice in combination with an opioid (fentanyl CRI) in animals with abdominal pain. Lidocaine is given as a bolus of 2 mg/kg (cats 0.5 mg/kg) followed by a CRI of 30 μg/kg/min (≈ 2 mg/kg/h) [Valverde *et al.*, 2004].

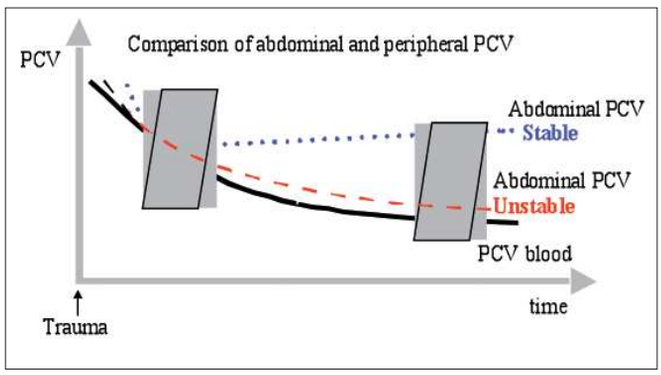

Figure 2: Comparison of venous and abdominal PCV. Abdominal bleeding is determined to have stopped when serial measurements of concurrent venous and abdominal PCV values show trends in opposite directions. After fluid resuscitation, the venous PCV value is suspected to decrease due to haemodilution. The abdominal fluid PCV is expected to decrease in a similar manner as the venous PCV if bleeding is continuing, whereas it is suspected to stay stable or increase due to fluid reabsorption if the bleeding has stopped

Arresting further haemorrhage

Arresting further haemorrhage requires normalization of haemostasis while maintaining adequate perfusion without hypertension. Abdominal counterpressure using an abdominal bandage may help to stop abdominal bleeding and increase survival [McAnulty and Smith, 1986]. A modification of this technique describes the incorporation of the pelvic limbs into the counterpressure bandage. Care must be taken to avoid the compartmentalization of blood in the pelvic limb vasculature and to avoid occluding the caudal abdominal vena cava [Crowe, 1988]. Our own experience shows that these bandages may be painful and difficult to safely apply. Abdominal counterpressure is contraindicated in patients with pelvic or femoral fractures, respiratory distress due to pneumothorax, pleural effusion or diaphragmatic rupture or with head trauma. Abdominal counterpressure can potentially lead to abdominal compartment syndrome with subsequent hypoperfusion of abdominal organs [Nieman *et al.*, 1983; Chang *et al.*, 1995] and are therefore rarely indicated in our opinion.

Medical versus surgical therapy

Depending on the cause and the progression of disease, surgery may be required to stop the bleeding. Several human studies and case reports have shown that diagnosis of haemoabdomen does not mandate immediate laparotomy [Goan *et al.*, 1998; Grisoni *et al.*, 1984; Hiatt *et al.*, 1990; Hoff *et al.*, 2002; Nagy *et al.*, 1995; Smith *et al.*, 1996]. Fluid resuscitation will lead to a decline in venous PCV, regardless of the presence of active or previous bleeding. In a retrospective veterinary study, 40% of the animals that underwent surgical intervention did not show active bleeding at the time of surgery [Mongil *et al.*, 1995].
The decision to treat a patient medically or surgically is often difficult. Haemodynamically unstable patients with signs of continued bleeding should undergo surgical intervention as soon as possible [Clarke *et al.*, 2002; Goan *et al.*, 1998; McKenney *et al.*, 1996; Smith *et al.*, 1996], as mortality in patients with severe abdominal bleeding that require surgical control was shown to increase with time [Clarke *et al.*, 2002]. Other problems that require surgical intervention should be ruled out as soon as possible [Mongil *et al.*, 1995, Prasse and Duncan, 1976].

However, patients with vague signs might pose a diagnostic challenge to the emergency clinician. Clinical, biochemical, haematological parameters, diagnostic imaging results as well as response to treatment have been suggested as aids to deciding if surgical intervention is necessary for the control of haemorrhage but did not prove to be helpful [Holt, 1978]. Clinical evaluation alone was shown to miss 59% of injuries in blunt trauma patients [Bivins and Sachatello, 1978; Smith *et al.*, 1996]. In a retrospective study of 28 dogs and cats with severe haemoabdomen, no clinical parameters that differentiated between the surgically and medically treated groups could be identified [Mongil *et al.*, 1995].

The initial venous PCV prior to fluid therapy has not been helpful in the evaluation of active bleeding [Snyder, 1998] and a normal PCV does not always indicate medical therapy is appropriate [Paradis *et al.*, 1997; Snyder, 1998].
Since neither repeated venous PCV measurements nor the determination of a single abdominal PCV identifies active bleeding, comparing abdominal fluid and serum PCV values continuously might be more useful in the selection of therapy [Sigrist and Spreng, 2007]. If abdominal bleeding has stopped, serial measurements of concurrent venous and abdominal PCV values will show trends in opposite directions, as venous blood will be diluted by fluid therapy while the abdominal PCV will not change. With continuous bleeding, the venous PCV will decrease and not stabilize with fluid therapy and the abdominal PCV will slowly decrease due to continuous bleeding with a lower venous PCV (see figure 2). This approach differs from an experimental study showing that using DPL, increasing PCV values in the DPL solution are associated with severe haemorrhage [Thomson

Figure 3: Splenic rupture.
This spleen has been traumatically transected leading to a severe haemoabdomen

et al., 1985]. Crowe (1993) recommends that increasing PCV values over time in DPL fluid is diagnostic for active abdominal haemorrhage.

Concurrent and serial measurements of the venous and abdominal PCV value were used in the case presented above. Initially, both values were similar. The venous PCV was lower at presentation due to fluid shifts from the interstitial to the intravascular space and initiation of fluid therapy. The stable abdominal PCV values over time despite declining venous PCV values are suggestive of an inactive haemoabdomen. Continued active haemorrhage would dilute the abdominal PCV. This method is only useful as long as the venous PCV decreases due to fluid therapy dilution and provided no blood products are transfused and is only applicable with a pure haemoabdomen without other fluid contamination.

Surgical Therapy

Patients that cannot be stabilized with fluid therapy are clearly candidates for a surgical approach. Rapid surgical exposure and a systematic approach are mandatory in order to localize and ligate the cause of haemorrhage [Herold 2008]. Hepatic or splenic ruptures are the most common causes of traumatic haemoabdomen; therefore these organs are primarily inspected (Figure 3). A modified Pringle maneuver has been described [Crowe and Devey, 1994] but has not been shown to be useful in the clinical setting.

If a large supply of blood products is available the surgical exploration can be performed more slowly. If at least 50% of the blood volume is available as blood products, the following approach may be used: rapid surgical exposure of the abdomen is followed by inspection of the spleen and control of potential splenic ruptures. Following splenic inspection, the abdominal fluid is suctioned in order to identify other bleeding sources such as a ruptured renal or hepatic vein [Feliciano and Moore, 2004]. This approach needs time and appropriate transfusion therapy. If unlimited transfusion therapy is not available or if large-volume haemorrhage is ongoing and the source is not immediately identified after inspection of spleen and liver, abdominal packing is recommended [Sharp and Lociero, 1992]. The abdomen is packed with laparotomy sponges or sterile towels. Once the packing material appears to have controlled major blood loss, the pads are methodically removed in a clockwise fashion starting at the caudal aspect of the abdomen and bleeding vessels identified and ligated. If the bleeding vessel cannot be identified very rapidly, the towels are left in place; the abdomen is temporarily closed, with re-exploration performed in 24-72 hours. This allows potential coagulopathies to be controlled and a chance for fluid resuscitation to occur.

Non-surgical therapy

In our experience, most traumatic haemoabdomen patients can be managed non-surgically. This has also been shown in various human studies [Goan *et al.*, 1998]. Other indications for surgery, such as uroabdomen or peritonitis should be ruled out. Non-surgical management includes maintaining perfusion and analgesia as described under stabilization. Animals are routinely monitored for perfusion deficits and PCV changes.

Monitoring

Both post-operative and non-surgically treated patients need intensive monitoring. Initially, monitoring of respiration, perfusion parameters such as heart rate, MM color and CRT, blood pressure and pulse quality may be necessary every 30 minutes. Subsequent monitoring depends on the haemodynamic changes. Tachycardia, pale mucous membranes and prolonged CRT are signs of hypovolaemic shock and need to be addressed immediately [de Laforcade 2008]. Anaemia leading to tachycardia and pale MM can be differentiated from hypovolaemia by a normal CRT.

Regular serial measurement of abdominal and venous PCV values (every 1-4 hours) is helpful in deciding if a surgical approach, blood products or colloids are indicated. Following aggressive fluid therapy, monitoring of coagulation parameters may be indicated to decrease the risk of ongoing or new bleeding.

Using the modified rule of 20 by Dr. Kirby allows identification and early correction of potential complications [Sigrist and Spreng, 2004].

Prognosis

One veterinary study showed that survival for animals with severe haemoabdomen is 67% for surgically treated patients and 75% for non-surgically managed patients [Mongil *et al.*, 1995]. Overall mortality in this study was 27 % [Mongil *et al.*, 1995]. Other veterinary studies are lacking and other trauma-related factors are important regarding prognosis of patients with traumatic haemoabdomen.

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